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=> e STI571/cn
E1            1     STI-X/CN
E2            1     STI50/CN
E3            0 --> STI571/CN
E4            1     STIA/CN
E5            1     STIALGIN/CN
E6            1     STIBA(5)FULLERANE-SB20-IH/CN
E7            1     STIBA(5)FULLERANE-SB20-IH, COMPD WITH PALLADIUM MOL. (PD12)
                    AND ANTIMONY ION (SB-3)/CN
E8            1     STIBA(5)FULLERANE-SB20-IH, COMPD. WITH KRYPTON AND MOL. NICK
                    EL (NI12) (1:1:1)/CN
E9            1     STIBA(5)FULLERANE-SB20-IH, COMPD. WITH KRYPTON AND MOL. PALL
                    ADIUNM (PD12) (1:1:1)/CN
E10           1     STIBABENZENE/CN
E11           1     STIBACETIN/CN
E12           1     STIBAMIN/CN

=> file caplus
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   0.46       0.67

FILE 'CAPLUS' ENTERED AT 14:26:25 ON 18 DEC 2008
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FILE COVERS 1907 - 18 Dec 2008  VOL 149 ISS 25
FILE LAST UPDATED: 17 Dec 2008  (20081217/ED)

Caplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2008.

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/legal/infopolicy.html

=> s STI571
L1         743 STI571

=> file hcaplus
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   3.56       4.23

FILE 'HCAPLUS' ENTERED AT 14:27:56 ON 18 DEC 2008
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FILE COVERS 1907 - 18 Dec 2008 VOL 149 ISS 25
FILE LAST UPDATED: 17 Dec 2008 (20081217/ED)

HCAplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

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=> s ischemia or reperfusion or myocardial or infarction of cardio?
         86597 ISCHEMIA
            76 ISCHEMIAS
         86612 ISCHEMIA
                 (ISCHEMIA OR ISCHEMIAS)
         37728 REPERFUSION
            56 REPERFUSIONS
         37738 REPERFUSION
                 (REPERFUSION OR REPERFUSIONS)
         80820 MYOCARDIAL
             2 MYOCARDIALS
         80821 MYOCARDIAL
                 (MYOCARDIAL OR MYOCARDIALS)
         46448 INFARCTION
          1258 INFARCTIONS
         46840 INFARCTION
                 (INFARCTION OR INFARCTIONS)
        183955 CARDIO?
           742 INFARCTION OF CARDIO?
                 (INFARCTION(1W)CARDIO?)
L2      148277 ISCHEMIA OR REPERFUSION OR MYOCARDIAL OR INFARCTION OF CARDIO?

=> s l1 and l2
           743 STI571
L3           2 L1 AND L2

=> d ibib abs hit 1-2

L3   ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2004:353588  HCAPLUS <<LOGINID::20081218>>
DOCUMENT NUMBER:         141:37106
TITLE:                   Inhibition of platelet-derived growth factor promotes
                         pericyte loss and angiogenesis in ischemic retinopathy
```

AUTHOR(S): Wilkinson-Berka, Jennifer L.; Babic, Sanja; De Gooyer, Tanyth; Stitt, Alan W.; Jaworski, Kassie; Ong, Leslie G. T.; Kelly, Darren J.; Gilbert, Richard E.
CORPORATE SOURCE: Department of Physiology, University of Melbourne, Parkville, Australia
SOURCE: American Journal of Pathology (2004), 164(4), 1263-1273
CODEN: AJPAA4; ISSN: 0002-9440
PUBLISHER: American Society for Investigative Pathology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We investigated whether inhibition of platelet-derived growth factor (PDGF) receptor tyrosine kinase activity would affect pericyte viability, vascular endothelial growth factor (VEGF)/vascular endothelial growth factor receptor-2 (VEGFR-2) expression and angiogenesis in a model of retinopathy of prematurity (ROP). ROP was induced in Sprague Dawley rats by exposure to 80% oxygen from postnatal (P) days 0 to 11 (with 3 h/day in room air), and then room air from P12-18 (angiogenesis period). Shams were neonatal rats in room air from P0-18. STI571, a potent inhibitor of PDGF receptor tyrosine kinase, was administered from P12-18 at 50 or 100 mg/kg/day i.p.. Electron microscopy revealed that pericytes in the inner retina of both sham and ROP rats appeared normal; however STI571 induced a selective pericyte and vascular smooth muscle degeneration. Immunolabeling for caspase-3 and α-smooth muscle cell actin in consecutive paraffin sections of retinas confirmed that these degenerating cells were apoptotic pericytes. In all groups, VEGF and VEGFR-2 gene expression was located in ganglion cells, the inner nuclear layer, and retinal pigment epithelium. ROP was associated with an increase in both VEGF and VEGFR-2 gene expression and blood vessel profiles in the inner retina compared to sham rats. STI571 at both doses increased VEGF and VEGFR-2 mRNA and exacerbated angiogenesis in ROP rats, and in sham rats at 100 mg/kg/day. In conclusion, PDGF is required for pericyte viability and the subsequent prevention of VEGF/VEGFR-2 overexpression and angiogenesis in ROP.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB We investigated whether inhibition of platelet-derived growth factor (PDGF) receptor tyrosine kinase activity would affect pericyte viability, vascular endothelial growth factor (VEGF)/vascular endothelial growth factor receptor-2 (VEGFR-2) expression and angiogenesis in a model of retinopathy of prematurity (ROP). ROP was induced in Sprague Dawley rats by exposure to 80% oxygen from postnatal (P) days 0 to 11 (with 3 h/day in room air), and then room air from P12-18 (angiogenesis period). Shams were neonatal rats in room air from P0-18. STI571, a potent inhibitor of PDGF receptor tyrosine kinase, was administered from P12-18 at 50 or 100 mg/kg/day i.p.. Electron microscopy revealed that pericytes in the inner retina of both sham and ROP rats appeared normal; however STI571 induced a selective pericyte and vascular smooth muscle degeneration. Immunolabeling for caspase-3 and α-smooth muscle cell actin in consecutive paraffin sections of retinas confirmed that these degenerating cells were apoptotic pericytes. In all groups, VEGF and VEGFR-2 gene expression was located in ganglion cells, the inner nuclear layer, and retinal pigment epithelium. ROP was associated with an increase in both VEGF and VEGFR-2 gene expression and blood vessel profiles in the inner retina compared to sham rats. STI571 at both doses increased VEGF and VEGFR-2 mRNA and exacerbated angiogenesis in ROP rats, and in sham rats at 100 mg/kg/day. In conclusion, PDGF is required for pericyte viability and the subsequent prevention of VEGF/VEGFR-2 overexpression and angiogenesis in ROP.

ST ischemia retinopathy angiogenesis pericyte PDGF VEGF VEGFR2
IT Angiogenesis
Apoptosis
Ischemia
(inhibition of platelet-derived growth factor promotes pericyte loss and angiogenesis in ischemic retinopathy)

L3 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:777593 HCAPLUS <<LOGINID::20081218>>
DOCUMENT NUMBER: 139:271094
TITLE: Inhibition of cell death responses induced by oxidative stress
INVENTOR(S): Kufe, Donald W.; Kaddurah-Daouk, Rima
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003080061	A1	20031002	WO 2003-US10112	20030320
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2479257	A1	20031002	CA 2003-2479257	20030320
AU 2003226209	A1	20031008	AU 2003-226209	20030320
AU 2003226209	B2	20081023		
EP 1487451	A1	20041222	EP 2003-745187	20030320
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 20060128720	A1	20060615	US 2005-518665	20051107
PRIORITY APPLN. INFO.:			US 2002-366410P P	20020321
			WO 2003-US10112 W	20030320

AB The invention provides methods of reducing or preventing oxidative stress-induced cell death by contacting a cell with a compound that inhibits the kinase activity and/or the mitochondrial translocation of c-Abl. The methods of the invention can be used to treat individuals individual diagnosed as having or being at risk of contracting a disorder characterized by excessive oxidative stress-induced cell death.
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT Nervous system, disease
(Huntington's chorea; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/ reperfusion injury in combination with other drugs)
IT Nervous system, disease
(amyotrophic lateral sclerosis; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of

c-Abl for treatment of neurol. disorders and ischemia/
reperfusion injury in combination with other drugs)
IT Membrane potential
(biol., mitochondrial; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/
reperfusion injury in combination with other drugs)
IT Medical goods
(catheters, drug delivery by; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/
reperfusion injury in combination with other drugs)
IT Disease, animal
(cellular, aging degeneration; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/
reperfusion injury in combination with other drugs)
IT Animal cell
(disease, aging degeneration; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/
reperfusion injury in combination with other drugs)
IT Heart, disease
(infarction; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion
injury in combination with other drugs)
IT Alzheimer's disease
Anti-Alzheimer's agents
Anti-inflammatory agents
Antiarthritics
Anticoagulants
Antioxidants
Antiparkinsonian agents
Apoptosis
Arthritis
Cell death
Coronary bypass surgery
Cytoprotective agents
Diagnosis
Dopamine agonists
Drug delivery systems
Drug interactions
Glutamate antagonists
Inflammation
Mitochondria
Multiple sclerosis
Nervous system, disease
Nervous system agents
Oxidative stress, biological
Parkinson's disease
Spinal muscular atrophy
Thrombolytics
Transplant and Transplantation
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in
combination with other drugs)
IT Reactive oxygen species

RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Growth factors, animal
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Drug delivery systems
(injections; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Reperfusion
(injury; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Biological transport
(intracellular; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Immunophilins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neuro-; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Cytoprotective agents
Nervous system agents
(neuroprotective agents; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Cell aging
(prevention; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Injury
(reperfusion; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Eye, disease
Inflammation
(retinitis pigmentosa; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT Brain, disease
(stroke; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment

of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 146838-19-9, Arg kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(C-Abl complexes; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 7722-84-1, Hydrogen peroxide, biological studies 7782-44-7D, Oxygen, reactive species
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 138238-67-2, c-Abl kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 52-49-3 321-64-2, Tacrine 616-91-1, N-Acetylcysteine 768-94-5, Amantadine 1744-22-5, Riluzole 14611-51-9, Selegiline 22260-51-1, Bromocriptine mesylate 57356-49-7D, derivs. 57828-26-9, Lipoic acid 66104-23-2, Pergolide mesylate 120014-06-4, Donepezil 220127-57-1, STI571
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 39391-18-9, Cyclooxygenase 122191-40-6, Interleukin 1β-converting enzyme 125978-95-2, Nitric oxide synthase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 59-92-7, Levodopa, biological studies
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mixture with carbidopa; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 28860-95-9, Carbidopa
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(mixture with levodopa; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)

IT 19771-63-2, Procysteine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(of neurol. disorders; inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/

reperfusion injury in combination with other drugs)

```
=> s tyrosine (3A) kinase
        175846 TYROSINE
          2806 TYROSINES
        176415 TYROSINE
                 (TYROSINE OR TYROSINES)
        337486 KINASE
         63244 KINASES
        347779 KINASE
                 (KINASE OR KINASES)
L4       53958 TYROSINE (3A) KINASE

=> s l1 and l3
           743 STI571
L5           2 L1 AND L3

=> d ibib 1-2

L5   ANSWER 1 OF 2  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2004:353588  HCAPLUS <<LOGINID::20081218>>
DOCUMENT NUMBER:         141:37106
TITLE:                   Inhibition of platelet-derived growth factor promotes
                         pericyte loss and angiogenesis in ischemic retinopathy
AUTHOR(S):               Wilkinson-Berka, Jennifer L.; Babic, Sanja; De Gooyer,
                         Tanyth; Stitt, Alan W.; Jaworski, Kassie; Ong, Leslie
                         G. T.; Kelly, Darren J.; Gilbert, Richard E.
CORPORATE SOURCE:        Department of Physiology, University of Melbourne,
                         Parkville, Australia
SOURCE:                  American Journal of Pathology (2004), 164(4),
                         1263-1273
                         CODEN: AJPAA4; ISSN: 0002-9440
PUBLISHER:               American Society for Investigative Pathology
DOCUMENT TYPE:           Journal
LANGUAGE:                English
REFERENCE COUNT:         45    THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 2 OF 2  HCAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2003:777593  HCAPLUS <<LOGINID::20081218>>
DOCUMENT NUMBER:         139:271094
TITLE:                   Inhibition of cell death responses induced by
                         oxidative stress
INVENTOR(S):             Kufe, Donald W.; Kaddurah-Daouk, Rima
PATENT ASSIGNEE(S):      Dana-Farber Cancer Institute, Inc., USA
SOURCE:                  PCT Int. Appl., 44 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2003080061        A1    20031002    WO 2003-US10112        20030320
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
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             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA,
             UG, US, UZ, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2479257           A1    20031002    CA 2003-2479257         20030320
     AU 2003226209        A1    20031008    AU 2003-226209          20030320
     AU 2003226209        B2    20081023
     EP 1487451           A1    20041222    EP 2003-745187          20030320
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     US 20060128720       A1    20060615    US 2005-518665          20051107
PRIORITY APPLN. INFO.:                     US 2002-366410P     P  20020321
                                           WO 2003-US10112     W  20030320
REFERENCE COUNT:         3     THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT



=> e STI 571/cn
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E2            1     STI 509-00/CN
E3            1 --> STI 571/CN
E4            1     STI TITANIA HICOAT Z 18-1000/CN
E5            1     STI ZEOLITES/CN
E6            1     STI-F 10G/CN
E7            1     STI-X/CN
E8            1     STI50/CN
E9            1     STIA/CN
E10           1     STIALGIN/CN
E11           1     STIBA(5)FULLERANE-SB20-IH/CN
E12           1     STIBA(5)FULLERANE-SB20-IH, COMPD WITH PALLADIUM MOL. (PD12)
                    AND ANTIMONY ION (SB-3)/CN

=> s e3
L10           1 "STI 571"/CN

=> d

L10  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2008 ACS on STN
RN   220127-57-1  REGISTRY
ED   Entered STN:  03 Mar 1999
CN   Benzamide, 4-[(4-methyl-1-piperazinyl)methyl]-N-[4-methyl-3-[[4-(3-
     pyridinyl)-2-pyrimidinyl]amino]phenyl]-, methanesulfonate (1:1)  (CA INDEX
     NAME)
OTHER CA INDEX NAMES:
CN   Benzamide, 4-[(4-methyl-1-piperazinyl)methyl]-N-[4-methyl-3-[[4-(3-
     pyridinyl)-2-pyrimidinyl]amino]phenyl]-, monomethanesulfonate (9CI)
OTHER NAMES:
CN   CGP 57148B
CN   Gleevac
CN   Gleevec
CN   Glivec
CN   Imatinib mesilate
CN   Imatinib mesylate
CN   STI 571
MF   C29 H31 N7 O . C H4 O3 S
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CI COM
SR CA
LC STN Files: ANABSTR, BIOSIS, BIOTECHNO, CA, CAPLUS, CBNB, CHEMCATS, CSCHEM, DDFU, DRUGU, EMBASE, HSDB*, IMSPATENTS, IMSRESEARCH, IPA, MRCK*, PATDPASPC, PHAR, PROUSDDR, PS, RTECS*, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)

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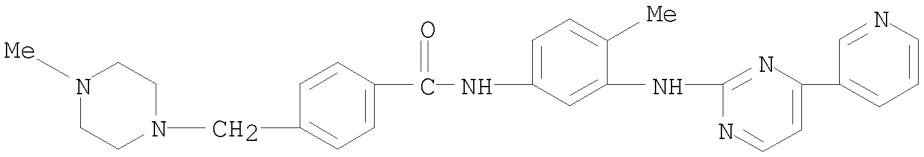

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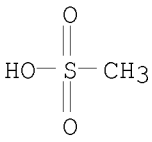

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

2445 REFERENCES IN FILE CA (1907 TO DATE)
27 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
2458 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> file caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	7.61	303.29

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-10.40

FILE 'CAPLUS' ENTERED AT 15:18:24 ON 21 DEC 2008

FILE COVERS 1907 - 21 Dec 2008 VOL 149 ISS 26
FILE LAST UPDATED: 19 Dec 2008 (20081219/ED)

Caplus now includes complete International Patent Classification (IPC)
reclassification data for the third quarter of 2008.

Effective October 17, 2005, revised CAS Information Use Policies apply.
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=> s l10
L11       2458 L10

=> s ischem? or reperfus? or stroke or myocardi? or infarct? or (organ (3a)
transplant?) or coronary
        102835 ISCHEM?
         38187 REPERFUS?
         40689 STROKE
          2687 STROKES
         42218 STROKE
                (STROKE OR STROKES)
        104191 MYOCARDI?
         51890 INFARCT?
        146856 ORGAN
        131675 ORGANS
        241015 ORGAN
                (ORGAN OR ORGANS)
        121493 TRANSPLANT?
          7528 ORGAN (3A) TRANSPLANT?
         82676 CORONARY
           269 CORONARIES
         82748 CORONARY
                (CORONARY OR CORONARIES)
L12     262969 ISCHEM? OR REPERFUS? OR STROKE OR MYOCARDI? OR INFARCT? OR (ORGA
               N (3A) TRANSPLANT?) OR CORONARY

=> s l11 and l12
L13        101 L11 AND L12

=> s l11(l) l12
L14          6 L11(L) L12

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L14  ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER:        2008:819689  CAPLUS <<LOGINID::20081221>>
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DOCUMENT NUMBER: 149:259029
TITLE: Activation of PDGF-CC by tissue plasminogen activator impairs blood-brain barrier integrity during ischemic stroke
AUTHOR(S): Su, Enming J.; Fredriksson, Linda; Geyer, Melissa; Folestad, Erika; Cale, Jacqueline; Andrae, Johanna; Gao, Yamei; Pietras, Kristian; Mann, Kris; Yepes, Manuel; Strickland, Dudley K.; Betsholtz, Christer; Eriksson, Ulf; Lawrence, Daniel A.
CORPORATE SOURCE: Department of Internal Medicine, Division of Cardiovascular Medicine, University of Michigan Medical School, Ann Arbor, MI, 48109-0644, USA
SOURCE: Nature Medicine (New York, NY, United States) (2008), 14(7), 731-737
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
AB TPA is a clot-buster used to treat stroke, but if it's given too late after stroke onset, it can cause complications like hemorrhage. Daniel Lawrence and his colleagues show that a US Food and Drug Administration-approved kinase inhibitor, Gleevec, can prevent this side effect, thereby extending tPA's therapeutic window. Thrombolytic treatment of ischemic stroke with tissue plasminogen activator (tPA) is markedly limited owing to concerns about hemorrhagic complications and the requirement that tPA be administered within 3 h of symptoms. Here we report that tPA activation of latent platelet-derived growth factor-CC (PDGF-CC) may explain these limitations. Intraventricular injection of tPA or active PDGF-CC, in the absence of ischemia, leads to significant increases in cerebrovascular permeability. In contrast, co-injection of neutralizing antibodies to PDGF-CC with tPA blocks this increased permeability, indicating that PDGF-CC is a downstream substrate of tPA within the neurovascular unit. These effects are mediated through activation of PDGF-α receptors (PDGFR-α) on perivascular astrocytes, and treatment of mice with the PDGFR-α antagonist imatinib after ischemic stroke reduces both cerebrovascular permeability and hemorrhagic complications associated with late administration of thrombolytic tPA. These data demonstrate that PDGF signaling regulates blood-brain barrier permeability and suggest potential new strategies for stroke treatment.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT 220127-57-1, Gleevec
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(activation of PDGF-CC by tissue plasminogen activator impairs blood-brain barrier integrity during ischemic stroke
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L14 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:819672 CAPLUS <<LOGINID::20081221>>
DOCUMENT NUMBER: 149:167005
TITLE: Imatinib buys time for brain after stroke
AUTHOR(S): Rieckmann, Peter
CORPORATE SOURCE: Division of Neurology, Brain Research Centre, University of British Columbia Hospital, Vancouver, BC, V6T 2B5, Can.
SOURCE: Nature Medicine (New York, NY, United States) (2008), 14(7), 712-713

CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The most effective drug to treat acute ischemic stroke, tissue plasminogen activator (tPA), must be applied within three hours after symptom onset because of the risk of hemorrhage and other complications such as neurotoxicity. The anticancer drug imatinib (Gleevec) may help overcome these limitations by counteracting the ability of tPA to increase the permeability of the blood-brain barrier.
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT 220127-57-1, Gleevec
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(imatinib buys time for brain after stroke)

L14 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:720754 CAPLUS <<LOGINID::20081221>>
DOCUMENT NUMBER: 149:258870
TITLE: Imatinib mesylate attenuates fibrosis in coxsackievirus b3-induced chronic myocarditis
AUTHOR(S): Leipner, Carola; Gruen, Katja; Mueller, Andreas; Buchdunger, Elisabeth; Borsi, Laura; Kosmehl, Hartwig; Berndt, Alexander; Janik, Tobias; Uecker, Andrea; Kiehntopf, Michael; Boehmer, Frank-D.
CORPORATE SOURCE: Institute of Virology, Medical Faculty, Friedrich Schiller University, Jena, Germany
SOURCE: Cardiovascular Research (2008), 79(1), 118-126
CODEN: CVREAU; ISSN: 0008-6363
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Aims: Coxsackievirus B3 (CVB3)-induced chronic myocarditis in mice is accompanied by severe fibrosis and by sustained elevation of platelet-derived growth factor (PDGF)-A, -B, and -C levels in the cardiac tissue. To test if PDGF stimulation of resident fibroblasts causally contributes to fibrosis, we employed inhibition of PDGF receptor signaling with the orally available kinase inhibitor Imatinib. Methods and results: Chronic myocarditis was induced by CVB3 infection of major histocompatibility complex (MHC) class II knockout (B6Aa0/Aa0) mice. The mice were treated with 100 mg/kg Imatinib or vehicle, resp., twice daily for 34 days. Expression of PDGF-C and of inflammatory cytokines were analyzed by semi-quant. RT-PCR. PDGFα receptor phosphorylation was detected by immunoblotting of cardiac tissue exts. and in situ by immunohistochem. Fibrosis formation was analyzed by Sirius-Red staining and hydroxyproline (HP) determination Fibronectin, and tenascin expression was analyzed by RT-PCR and immunohistochem. Matrix metalloproteinase (MMP) activity was assessed with collagen, synthetic peptides, and gelatine as substrates. Imatinib significantly inhibited the myocarditis-related PDGFα receptor activation in the heart tissue. The virus titers in the hearts, inflammatory infiltrations, and elevated PDGF levels were unaffected by the Imatinib treatment. A significant attenuation of fibrosis occurred in Imatinib-treated animals. The Sirius Red-stained fibrotic area was reduced from 5.30 ± 0.50 to 3.21 ± 0.35%, and the HP content was reduced from 362 ± 43 to 238 ± 32 μMol/10 mg dry weight vs. 190 ± 27 in uninfected controls. The expression of fibronectin, EIIIA+ fibronectin, and tenascin C were likewise reduced. The diminished matrix protein deposition was not caused by elevated MMP

activity, since MMP activity was not changed or even reduced under Imatinib. Conclusion: The data suggest a causal role for elevated PDGF expression and PDGF receptor activity in the pathogenesis of cardiac fibrosis.

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

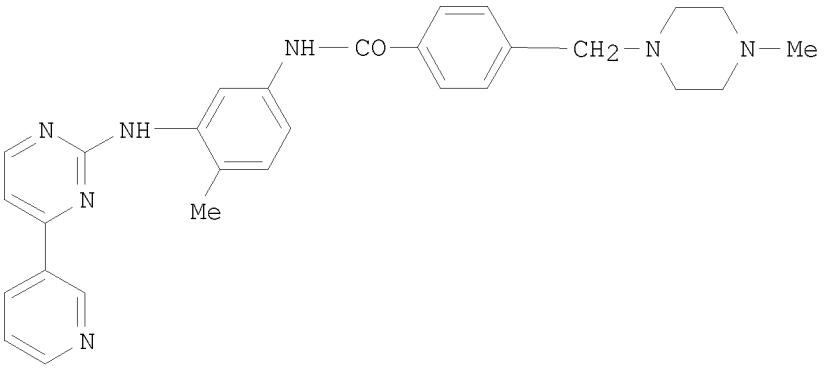
IT 220127-57-1, Imatinib mesylate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(imatinib mesylate attenuates fibrosis in coxsackievirus b3-induced chronic myocarditis)

L14 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:696750 CAPLUS <<LOGINID::20081221>>
DOCUMENT NUMBER: 143:166661
TITLE: Use of PDGF receptor tyrosine kinase (PDGF-R TK) inhibitors for the treatment of myocarditis and its complications
INVENTOR(S): Leipner, Carola; Boehmer, Frank-Dietmar; Gruen, Katja; Shetty, Suraj Shivappa; Massimini, Giorgio
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005070432	A1	20050804	WO 2005-EP749	20050126

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: GB 2004-1761 A 20040127
GI



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AB The invention discloses the use of a PDGF-R TK inhibitor, e.g. I, or a pharmaceutically acceptable salt thereof, for the manufacture of pharmaceutical compns. for the treatment of myocarditis and/or its complications.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT 71897-07-9, AG1295 146535-11-7, AG1296 152459-95-5 190726-45-5, KI 6783 194409-57-9, RP 1776 200706-56-5D, derivs. 205255-11-4, KN 1022 205256-55-9, CT52923 214983-11-6, PD 170262 220064-45-9, GFB 111 220127-57-1 252916-29-3, SU6668 339184-09-7, CDP 860 343787-29-1, CP 673451 387867-13-2, MLN 518 557795-19-4, SU 11248 692737-80-7, CHIR 258 777080-36-1, AG 13736 804551-01-7, SU 102 (kinase inhibitor) 804551-02-8, RPR 101511A 860792-92-3, Zvegf 3
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(PDGF receptor tyrosine kinase inhibitors for treatment of myocarditis and complications)

L14 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:836581 CAPLUS <<LOGINID::20081221>>
DOCUMENT NUMBER: 139:345919
TITLE: Regeneration of endogenous myocardial tissue by induction of neovascularization
INVENTOR(S): Itescu, Silviu
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 51 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030199464	A1	20031023	US 2002-128738	20020423
CA 2482996	A1	20031106	CA 2003-2482996	20030423
WO 2003090512	A2	20031106	WO 2003-US12768	20030423
WO 2003090512	A3	20041104		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003231090	A1	20031110	AU 2003-231090	20030423
EP 1501852	A2	20050202	EP 2003-724217	20030423
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1662548	A	20050831	CN 2003-814715	20030423
CN 100379751	C	20080409		
JP 2005534290	T	20051117	JP 2003-587162	20030423
US 20040247564	A1	20041209	US 2003-693480	20031023
US 20050233992	A1	20051020	US 2005-512518	20050615
US 20070172467	A1	20070726	US 2006-648769	20061229

PRIORITY APPLN. INFO.: US 2002-128738 A 20020423
WO 2003-US12768 W 20030423
US 2005-512518 A1 20050615

AB This invention provides a method of treating a disorder of a subject's heart involving loss of cardiomyocytes which comprises administering to the subject an amount of an agent effective to cause cardiomyocyte proliferation within the subject's heart to thereby treat the disorder. This invention further provides the instant method wherein the agent is human endothelial progenitor cells. This invention also provides methods of determining the susceptibility of a cardiomyocyte in a subject to apoptosis.

IT 220127-57-1, STI-571
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(regeneration of endogenous myocardial tissue by induction of neovascularization using human endothelial progenitor cells and inhibitor of c-Abl tyrosine kinase activation)

L14 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:777593 CAPLUS <<LOGINID::20081221>>
DOCUMENT NUMBER: 139:271094
TITLE: Inhibition of cell death responses induced by oxidative stress
INVENTOR(S): Kufe, Donald W.; Kaddurah-Daouk, Rima
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., USA
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003080061	A1	20031002	WO 2003-US10112	20030320
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2479257	A1	20031002	CA 2003-2479257	20030320
AU 2003226209	A1	20031008	AU 2003-226209	20030320
AU 2003226209	B2	20081023		
EP 1487451	A1	20041222	EP 2003-745187	20030320
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 20060128720	A1	20060615	US 2005-518665	20051107

PRIORITY APPLN. INFO.: US 2002-366410P P 20020321
WO 2003-US10112 W 20030320

AB The invention provides methods of reducing or preventing oxidative stress-induced cell death by contacting a cell with a compound that inhibits the kinase activity and/or the mitochondrial translocation of c-Abl. The methods of the invention can be used to treat individuals individual diagnosed as having or being at risk of contracting a disorder characterized by excessive oxidative stress-induced cell death.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT 52-49-3 321-64-2, Tacrine 616-91-1, N-Acetylcysteine 768-94-5, Amantadine 1744-22-5, Riluzole 14611-51-9, Selegiline 22260-51-1, Bromocriptine mesylate 57356-49-7D, derivs. 57828-26-9, Lipoic acid 66104-23-2, Pergolide mesylate 120014-06-4, Donepezil 220127-57-1, STI571
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(inhibition of cell death responses to oxidative stress by inhibiting kinase or mitochondrial translocation of c-Abl for treatment of neurol. disorders and ischemia/reperfusion injury in combination with other drugs)